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Research Report

Encoding strategy mediates the effect of electrical stimulation over posterior parietal cortex on visual short-term memory

Sisi Wang [a,b,c,d], Sirawaj Itthipuripat [d,e,f,**,1] and Yixuan Ku [a,b,c,g,*,1]

[a] Guangdong Provincial Key Laboratory of Social Cognitive Neuroscience and Mental Health, Department of Psychology, Sun Yat-Sen University, Guangzhou, China
[b] Peng Cheng Laboratory, Shenzhen, China
[c] Shanghai Key Laboratory of Brain Functional Genomics, Shanghai Changning-ECNU Mental Health Center, School of Psychology and Cognitive Science, East China Normal University, Shanghai, China
[d] Department of Psychology, Vanderbilt University, Nashville, TN, USA
[e] Learning Institute, King Mongkut's University of Technology Thonburi, Bangkok, Thailand
[f] Futuristic Research in Enigmatic Aesthetics Knowledge Laboratory, King Mongkut's University of Technology Thonburi, Bangkok, Thailand
[g] NYU-ECNU Institute of Brain and Cognitive Science, NYU Shanghai and Collaborative Innovation Center for Brain Science, Shanghai, China

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ABSTRACT

Over past decades, converging neuroimaging and electrophysiological findings have suggested a crucial role of posterior parietal cortex (PPC) in supporting the storage capacity of visual short-term memory (VSTM). Moreover, a few recent studies have shown that electrical stimulation over PPC can enhance VSTM capacity, making it a promising method for improving VSTM function. However, the reliability of these results is still in question because null findings have also been observed. Among studies that reported significant effects, some found increased VSTM capacity only in people with low capacity. Here, we hypothesized that subjects' encoding strategy might be a key source of these variable results. To directly test this hypothesis, we stimulated PPC using transcranial direct-current stimulation (tDCS) in male and female human subjects instructed to employ different encoding strategies during a VSTM recall task. We found that VSTM capacity was higher in subjects who were instructed to remember all items in the supra-capacity array of visual stimuli (i.e., the remember-all group), compared to subjects who were told to focus on a subset of these stimuli (i.e., the remember-subset group). As predicted, anodal tDCS over PPC significantly enhanced VSTM capacity only in the remember-subset group, but not in the remember-all group. Additionally, no effect of encoding strategy or its interaction with

* *Corresponding author*. Department of Psychology, Sun Yat-Sen University, Guangzhou, China.
** *Corresponding author*. Learning Institute, King Mongkut's University of Technology Thonburi, Bangkok, Thailand.
E-mail addresses: wangsisi2017@gmail.com (S. Wang), itthipuripat.sirawaj@gmail.com (S. Itthipuripat), kuyixuan@mail.sysu.edu.cn (Y. Ku).
[1] Equal contributions as co-corresponding authors.
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electrical stimulation was found on VSTM precision. Together, these results suggest that encoding strategy has a selective influence on VSTM capacity and this influence of encoding strategy mediates the effect of electrical stimulation over PPC on VSTM function.

1. Introduction

Visual short-term memory (VSTM) provides an active storage for visual information to be temporally stored so that it could be manipulated and readily used to guide behaviors (Baddeley & Hitch, 1974; Cowan, 2001). Over past decades, converging evidence has suggested an essential role of the posterior parietal cortex (PPC) in supporting VSTM storage. For example, human fMRI and EEG studies have shown that PPC activity tracks VSTM capacity or the number of items maintained in VSTM (Ikkai, McCollough, & Vogel, 2010; Luria, Balaban, Awh, & Vogel, 2016; Todd & Marois, 2004, 2005; Vogel & Machizawa, 2004; Xu & Chun, 2006). Moreover, recent studies have shown that transcranial direct-current stimulation (tDCS) over human PPC can cause changes in VSTM function, making it a promising non-invasive method for improving VSTM function (Heimrath, Sandmann, Becke, Müller, & Zaehle, 2012; Hsu, Tseng, Liang, Cheng, & Juan, 2014; Jones & Berryhill, 2012; Li et al., 2017; Tseng et al., 2012; Wang, Itthipuripat, & Ku, 2019). Recently, we have also found that stimulating the PPC selectively improves VSTM capacity compared to stimulating the prefrontal cortex (PFC), suggesting that the stimulation-induced enhancement of VSTM function is specific to the PPC and it is not due to global excitability that is not specific to this brain region (Wang et al., 2019).

While positive evidence may suggest that the PPC has a casual role in supporting VSTM function, recent meta-analyses of tDCS studies have suggested that the reported significant effects of PPC stimulation on VSTM function are unreliable (Horvath, Forte, & Carter, 2015; Mancuso, Ilieva, Hamilton, & Farah, 2016). While some empirical studies reported equally robust stimulation effects across individual subjects (Li et al., 2017; Wang et al., 2019), some revealed a selective stimulation effect for a specific group of individuals (e.g., individuals with low capacity and older adults) and null findings (e.g., Arciniega, Gözenman, Jones, Stephens, & Berryhill, 2018; Hsu et al., 2014; Robison, McGuirk, & Unsworth, 2017; Tseng et al., 2012).

Here, we hypothesized that differences in subjects' encoding strategy might be a key source of these variable results. In line with our theory, it has been suggested that the individual differences in VSTM capacity might be driven by individual differences in encoding strategy employed during VSTM tasks (Cusack, Lehmann, Veldsman, & Mitchell, 2009; Linke, Vicente-Grabovetsky, Mitchell, & Cusack, 2011; Bengson & Luck, 2016; Donkin, Kary, Tahir, & Taylor, 2016; Atkinson, Baddeley, & Allen, 2018). For example, one recent study has demonstrated that instructing subjects to use different encoding strategies could directly impact their VSTM capacity (Bengson & Luck, 2016). By telling subjects to either remember all of the items in the supra-capacity stimulus array (i.e., the remember-all group) or only focus on a subset of these items (i.e., the remember-subset group), they found that the remember-all group had significantly higher VSTM capacity than the remember-subset group. Derived from this finding, we predicted that the remember-all strategy might place subjects in the performance level that reaches the upper limit of VSTM capacity and PPC stimulation in these subjects could no longer improve their VSTM capacity. On the other hand, the performance of subjects who employ the remember-subset strategy is lower than the VSTM upper limit. Thus, they could benefit from PPC stimulation.

To test these predictions, we adapted the method developed by Bengson and Luck (2016), where we manipulated encoding strategies across two subject groups in a VSTM recall task via different instructions: remember-all and remember-subset groups. Across two days, subjects in both groups received either PPC or sham stimulation for 15 min before performing the VSTM recall task. Consistent with Bengson and Luck (2016), we found that the remember-all group had higher VSTM capacity compared to the remember-subset group. Importantly, PPC stimulation significantly enhanced VSTM capacity only in the remember-subset group but not in the remember-all group, and this stimulation-induced enhancement reached the similar performance level in the remember-all group. Taken together, these findings suggest that the previously observed discrepancy in the PPC stimulation effects on VSTM performance could be driven by individual differences in VSTM capacity caused by difference in encoding strategy.

2. Materials and methods

We report how we determined our sample size, all data exclusions, all inclusion/exclusion criteria, whether inclusion/exclusion criteria were established prior to data analysis, all manipulations, and all measures in the study.

2.1. *Participants*

Forty human adults were recruited from East China Normal University (ECNU) [31 females, age range = 18–26, mean age (SD) = 22.05 (2.15), all right-handed]. All participants had normal or corrected-to-normal vision, no metallic implant, and no history of any neurological/psychiatric disease. Prior to their participation, all subjects provided written informed consent as required by the ethics committee at ECNU. They were randomly assigned to different subject groups: the remember-all and remember-subset groups with 20 subjects for each group. The sample size of 20 subjects for each

strategy group is within the typical range of tDCS studies using similar task designs and multisession approaches (10–20 subjects in Tseng et al., 2012; Heinen et al., 2016; Reinhart and Woodman, 2014, 2015, 2016).

2.2. Stimuli and experimental paradigm

2.2.1. Visuospatial working memory span task

For each day prior to receiving electrical stimulation, participants also completed a visuospatial working memory (WM) span task, proven to provide a reliable and valid behavioral measure of WM capacity (see review Conway et al., 2005). Fig. 1 shows the visuospatial WM span task in the current experiment. In this task, on each trial green squares appeared one-by-one across 3–9 different locations in a random sequence (without appearing at the same location over once). These green squares appeared in any of 25 locations in a 5 × 5 white square matrix (1 cm width x 1 cm height for each location, 2 cm apart) with 800 msec inter-stimulus interval. Participants were asked to remember the locations of the green squares in sequence and hold these memories during a blank delay of 500 msec. At the end of each trial, the test display containing 25 white square probes appeared on the screen. They had to recall the locations of the green squares on the screen by clicking on the white squares in sequence. In order to measure the visuospatial WM span, we must know the set size at which subjects started to perform poorly. Thus, we used an adaptive task, in which the experiment started from lower to higher set sizes (from set size 3 to 9, easy to difficult). If participants answered correctly more than one of three consecutive trials, set size would increase in one incremental step and the experiment would end if subjects answered correctly one or none of the three consecutive trials. We then subtracted .5 (half step) from the last set size to determine the behavioral threshold at which subjects started to perform poorly. This behavioral threshold was taken as their visuospatial WM span. For example, if a participant ends at set size 6, his/her visuospatial WM span is 5.5.

The experimental paradigm codes, raw data, summary data, and data analysis methods for the visuospatial WM span task are publicly available at the Open Science Framework. Readers can visit https://osf.io/ugdh9/ to get these materials.

2.2.2. Continuous recall task

We presented all stimuli on a 24″ Dell monitor (Refreshing rate: 60 Hz) running MATLAB (R2011b) (Mathworks Inc., Natick, MA) and the Psychophysics Toolbox (version 3.0.12; Brainard, 1997; Pelli, 1997). Subjects were seated 60 cm from the monitor with a gray background (RGB: 192 192 192) in a sound-attenuated room. Fig. 2.a depicts a schematic of the experimental paradigm. Each trial started with a white fixation cross (RGB: 255 255 255) in the center of the screen. Then a stimulus array containing 4, 6 or 8 different oriented black bars (2.0° × .3° visual angle; RGB: 0 0 0) appeared on the screen for 200 msec, followed by a delay of a blank screen for 1000 msec. Individual bars located at the eccentricity of 6° visual angle with a center distance of at least 30° between two adjacent bars. We randomly chose the orientations of these bars from 10 to 170° polar angle, which were at least 10° apart from one another. A black circular probe (2° visual angle inner diameters, .3° visual angle thickness) then pseudo-randomly appeared at one of the previously presented stimulus locations. Participants were told to report the orientation of the remembered stimulus at the probed location by clicking on the black circular probe using a mouse as precisely as possible (on either side of the bar to produce a virtual bar across the center of the circle). There was no response deadline. Inter-trial intervals were jittered from 500 msec to 1000 msec. The length or the width of the stimuli in the current experiment were calculated by using the tangent of a given visual angle multipling the distance between participant's eye and the screen as well as the length or the width of the screen in pixel divided by the length or the width of the screen in centimeter.

2.3. Experimental procedures

On the first day, participants first completed two practice blocks of the VSTM recall task during the strategy learning session, which included 50 trials of set size 4 and 50 trials of set size 6, respectively. Based on their assigned groups,

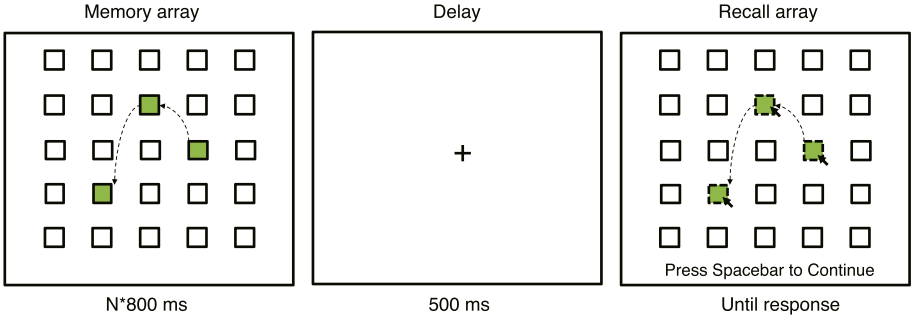

Fig. 1 – The visuospatial working memory (WM) span task. This is an illustration of the WM span task in set size 3 condition. The dashed arrows in the memory array (not physically presented) indicate the sequence of the to-be-remembered items (i.e., green squares). N represents the number of locations of the green squares (varied from 3 to 9). After the blank delay, participants recalled the locations of the green squares in sequence by using a mouse to click on the white squares in the correct order.

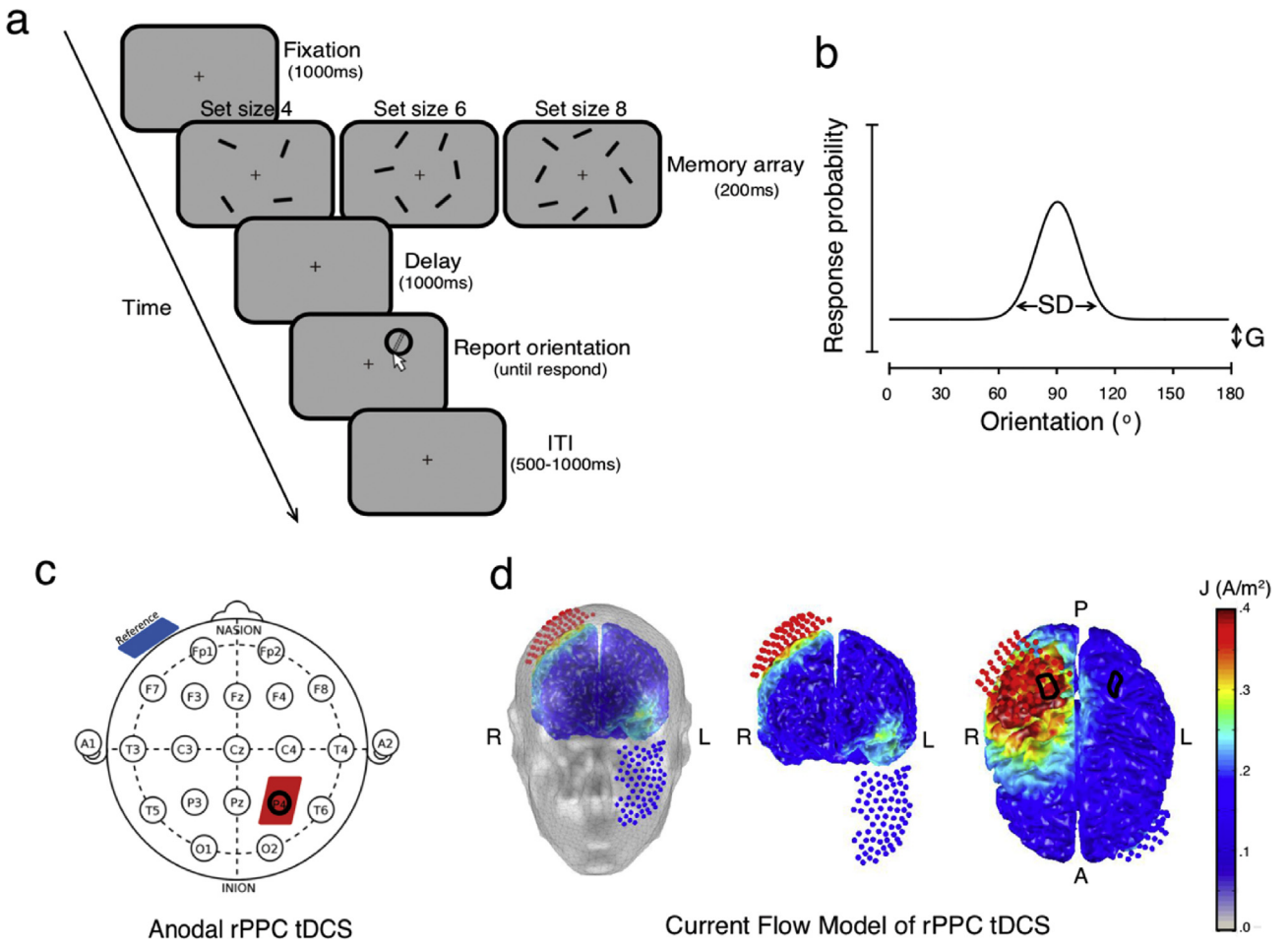

Fig. 2 – Experimental procedure and tDCS set-up. (a) Schematic diagrams of the VSTM recall task. Each trial started with an array of 4, 6, or 8 randomly oriented bars, followed by a 1000 msec delay when subjects hold these items in mind. After the delay, a probe circle appeared at one of the remembered locations, and subjects reported the orientation of the bar previously presented at that location by a mouse click. The unfilled bar in the probe circle was an illustration of the reported orientation and did not psychically appear in the real task. (b) Standard mixture model of recall performance. The solid black line comprises a mixture of two trials types: trials where items are remembered and trials in which items are not remembered. Parameter G represents the height of the uniform distribution and SD illustrates the standard deviation or the width of the recall error distribution. (c) The placement of tDCS electrodes. A red patch represents the anodal electrode (P4) and a blue patch represents the reference electrode (left cheek). (d) The current density distribution for anodal PPC tDCS. The current density J is defined as the electric current per unit of cross-sectional area (Heald & Marion, 1995). A/m^2 is a unit of J, which stands for amp per meter squared. The left panel shows electrode locations on the scalp surface and the contralateral cheek (red and blue dots for the anodal and the reference electrodes, respectively) as well as the cortical current density distribution from the front view. The middle panel shows the cortical current density distribution from the front view, and the right panel shows the cortical current density distribution from the overhead view, the areas within black circles represent sub-areas that show load-dependent effect in fMRI activity during a variant of the VSTM tasks (Magen, Emmanouil, McMains, Kastner, & Treisman, 2009; Todd & Marois, 2004; Xu & Chun, 2006). 'L', 'R', 'A', and 'P' stand for left, right, anterior, and posterior, respectively.

individual participants were given different task instructions so that they deployed different encoding strategies (see Bengson & Luck, 2016). The instruction for remember-all group was "Try to remember the entire display at the same time, no matter how many items are presented". The instruction for remember-subset group was: "If you can't remember the entire array, focus on a subset and try to remember them as precisely as possible". The instructions were shown to participants at the beginning of every block to remind them to use the assigned memory strategy. Then they received 15 min of PPC stimulation or sham stimulation. Immediately after the stimulation protocol ended, they completed six blocks of the VSTM recall task. Memory set size was varied across six blocks of 50 trials and block types were pseudo-randomized. Each subject completed 100 trials for each memory set size, 300 trials in total for each session day. No part of the study procedures was pre-registered prior to the research being conducted.

We chose the between-groups design for task strategy here for two main reasons. First, we would like to follow closely the experimental design introduced by Bengson and Luck (2016) in order to replicate their findings since their results have not been replicated at least to our knowledge. The fact that we could replicate their findings using a continuous recall task suggests that this between-groups design successfully taps into the interaction between the encoding strategy and VSTM functions (see the Results). Second, different encoding strategies require training. As described in the method section and according to the protocol developed by Bengson and Luck (2016), subjects had to undergo training sessions to ensure that they would implement a given encoding strategy during the actual recall task after they received PPC/sham stimulation. Using the within-subjects instead of between-subjects design for manipulating the encoding strategy here might have several disadvantages. For example, subjects would have been aware of both strategies and might have used only one strategy that they preferred. Moreover, there was no guarantee that they would not switch between the two strategies on a trial-by-trial basis. Furthermore, adding encoding strategy as another within-subjects factor would have cut the number of trials for each condition in half, and this would have worsened the model fitting of the response error distributions because stable fit parameters require a lot of trials from the continuous recall task. Considering all of these issues related to the within-subjects design, we decided to use the between-subjects design, following Bengson and Luck (2016).

2.4. Data analysis

2.4.1. Circular standard deviation

First, to examine how participants' responses deviated from the actual presented orientation of the remembered stimulus, the circular standard deviation of the recall errors (the degree difference between the participants' reported orientation and the actual presented orientation) in different conditions was computed using a Circular Statistics Toolbox for Matlab (Berens, 2009). Higher circular standard deviation indicates higher raw recall errors or lower memory fidelity. Two participants (one from each group) were excluded from the main analysis because of their extremely poor memory performance; their circular standard deviation of the recall errors calculated from the degree difference between the presented and reported orientations exceeded 2 SDs of all subjects in both groups, resulting in 19 participants for each group in the final analysis.

2.4.2. Model comparison

The circular standard deviation can only reflect the fidelity of the responses in general. To dissociate how many items are maintained and how precisely these items are presented in VSTM from the recall errors, the recall errors were then fitted into computational model. Two popular models that have been used to fit the VSTM response error include the Standard Mixture model (Zhang & Luck, 2008) and Swap model (Bays & Husain, 2008), which operate under different assumptions. The Standard Mixture model assumes that VSTM contains limited number of discrete slots (Zhang & Luck, 2008). In contrast, the Swap model assumes that VSTM is a system with flexible resources which does not have a capacity limit (Bays & Husain, 2008). Although our studies did not specifically aim to test whether the effects of tDCS on VSTM functions are better supported by one of these models/theories, we think it is a good practice to compare which of these models better described the data to make a contact to the larger body of VSTM literature and whether they yielded consistent results. Overall, we found that the Standard Mixture model generally fits our data better than the Swap model. We compared the Standard Mixture model and the Swap model by comparing the Akaike Information Criterion (AIC) and the Bayesian Information Criterion (BIC) values across these two models. For AIC, the Standard Mixture model outperformed the Swap model and provided better fits for 18 out of 38 subjects while the Swap model fitted better for 11 out of 38 subjects, and 9 participants with equal performance of the two models. For BIC, the Standard Mixture model provided better fits for 29 out of 38 subjects while the Swap model fitted better for 3 out of 38 subjects, and 6 participants with equal performance of the two models. Therefore, the main analysis focused on the model parameters (G and SD) obtained from the Standard Mixture model, although the overall results were qualitatively consistent across the two modeling approaches.

2.4.3. Modeling response errors

According to the Standard Mixture model, response deviations from the actual orientation reflect a mixture of trials where the probed bars were remembered and trials where observers guessed randomly (Zhang & Luck, 2008, 2009, 2011). As shown in Fig. 2.b, the distribution of recall errors consists of a mixture of a von Mises distribution (the remembered representation similar to circular Gaussian distribution around the correct orientation) and a uniform distribution (random guesses) (Zhang & Luck, 2008). To determine VSTM capacity and precision of each condition, we fitted the distribution of recall errors with the Standard Mixture model using a Maximum Likelihood Estimation (MLE) in the Memtoolbox (Suchow, Brady, Fougnie, & Alvarez, 2013) in Matlab (R2011b) (Mathworks Inc., Natick, MA). Here, two parameters, G and SD, can be extracted for each experimental condition and each subject. G reflects the height of the uniform distribution or the guessing probability. SD represents the width of the distribution of response errors on trials where the probed item was remembered. Next, we converted G to the probability that the probed item was present in memory (Pm = 1-G). VSTM capacity (K) was then estimated by multiplying Pm with memory set size. In addition, VSTM precision was obtained from computing the inverse of SD (i.e., SD^{-1}). Thus, the smaller SD is, the higher WM precision is.

2.5. tDCS set-up

We delivered tDCS using a battery driven constant current stimulator (Eldith, NeuroConn GmbH, Germany) with a pair of rubber electrodes in 5 × 7 cm^2 saline-soaked synthetic sponges. As shown in Fig. 2.c, we placed the anodal electrode over P4 according to the International 10–20 system following previous studies (Berryhill, Wencil, Branch Coslett, & Olson, 2010; Hsu et al., 2014; Jones & Berryhill, 2012; Li et al., 2017; Sandrini, Fertonani, Cohen, & Miniussi, 2012; Tseng et al.,

2012; Wang et al., 2019) and the reference electrode over the left cheek (Berryhill et al., 2010; Hsu et al., 2014; Jones & Berryhill, 2012; Li et al., 2017; Tseng et al., 2012; Wang et al., 2019). During this anodal PPC stimulation, we passed a constant current of 2.0 mA continuously for 15 min, with a linear fade in and fade out of 20 sec. For sham stimulation, the electrode placements were identical to the PPC stimulation protocol, but the duration of an actual stimulation in the sham condition lasted for only 30 sec, subjects received no stimulation for the rest 14mins and 30 sec with the electrodes remained. This ensured that in the sham condition participants experienced similar itches that might occur and recede over the first few seconds of an active stimulation. The current study was a single-blinded experiment. Individual participants underwent the PPC stimulation and the sham stimulation on two different days without being told what kind of stimulation they were accepting, separated by at least 48 hours to minimize potential carryover stimulation effects. In addition, we counterbalanced the order of stimulation types across participants.

To prevent confounds from subjects' awareness of differences in stimulation protocols which could interfere with their behavioral performance, we told subjects that they would receive stimulation on both days, and they might feel tingling and/or itches on both days without telling them in detail about our sham and active stimulation protocols (c.f. Kessler, Turkeltaub, Benson, & Hamilton, 2012). We also told subjects prior to the experiment that the stimulation might lead to better or worse performance so that they could not adjust their behavior in a specific direction based on guessing by the sensation from the stimulation. Note that we told them at the end of the second day about sham and active stimulation in detail.

2.6. *Current flow model*

For visualizing the current density distribution of our tDCS protocol, we used COMETS (a Matlab toolbox, written by Jung, Kim, & Im, 2013) to simulate local electric fields under tDCS. The current density model of the anodal PPC stimulation in the present study was shown in Fig. 2.d. COMETS used the head model extracted from standard Montreal Neurological Institute (MNI) brain atlas (Collins, Neelin, Peters, & Evans, 1994) to simulate the electric fields. A three-layer boundary element method (BEM) consisting of the scalp, skull boundaries and cerebrospinal fluid, as well as cortical surface model extracted from MRI T1 images of standard brain atlas via CURRY6 was used in this head model. Conductivity values for the scalp, skull and cerebral spinal fluid (CSF) were set as .22, .014 and 1.79 (S/m), respectively (Haueisen, Ramon, Eiselt, Brauer, & Nowak, 1997). The first step to simulate the electric fields was to set the bipolar electrode configurations (position, size and intensity) on the head model (position: anodal-P4 channel, reference-left cheek; size: 5 × 7cm^2; intensity: 2.0 mA in the current experiment). Then, the parameters were applied to a 3D finite element modeling (FEM) method based on electrostatic Laplace equation to analyze the current density inside the human head produced by the current tDCS setup (Jung et al., 2013). The current density J (A/m^2) is defined as the electric current per unit of cross-sectional area. It can be calculated by the following formula: J = I/A, where I is the electric current, and A is the active area of the electrode (Heald & Marion, 1995). Therefore, higher J values represent larger current density passing through the stimulated area, and its unit A/m^2 stands for amp per meter squared.

2.7. *Statistical analysis*

The current experiment is a mixed design, with a between-subjects factor of task strategy (remember-all and remember-subset) and two within-subjects factors of set size (4, 6 and 8) and stimulation type (PPC stimulation and sham stimulation). All statistical analyses were performed using SPSS 19.0 (IBM Inc.). First, we examined the effects of task strategy and set size as well as their interaction on the sham stimulation data to replicate the findings previously reported by Bengson and Luck (2016). We performed mixed ANOVAs with the between-subjects factor of task strategy (remember-all and remember-subset) and the within-subjects factor of set size (4, 6 and 8) on VSTM capacity (K) and precision (SD^{-1}) obtained from the sham stimulation condition. Since there was a significant interaction between task strategy and set size on K, we performed post-hoc independent sample *t*-tests to examine the effect of task strategy on K for each of the 3 set sizes (2-tailed), and multiple comparisons were corrected using the Holm-Bonferroni method (Ludbrook, 1998). Next, we examined the effect of PPC stimulation on K and SD^{-1}. To do so, we performed mixed ANOVAs with the between-subjects factor of task strategy and other two within-subjects factors, including set size and stimulation type (PPC stimulation and sham stimulation) on parameters K and SD^{-1}. Since there was a significant three-way interaction between these factors on K, we performed a follow-up repeated measures ANOVA with the within-subjects factors of set size and stimulation type separately for each subject group. Because there was a significant interaction between stimulation type and set size in the remember-subset group, we further performed post-hoc paired sample *t*-tests to test the stimulation effects on K separately for each of the 3 set sizes (2-tailed), and multiple comparisons were corrected using the Holm-Bonferroni method. Based on a significant stimulation effect in set size 8 in the remember-subset group, we ran another independent sample *t*-test to see if this stimulation effect in set size 8 in this group was higher than the remember-all group, where no significant stimulation effect was found. No part of the study analyses was pre-registered prior to the research being conducted.

The experimental paradigm codes, raw data, summary data, and data analysis codes for the VSTM recall task are publicly available at the Open Science Framework. Readers can visit https://osf.io/ugdh9/to get these materials.

3. Results

3.1. *No difference in visuospatial working memory span between two strategy groups*

To exclude the possibility that any potential effect of strategy or tDCS on VSTM performance could be due to the difference

of VSTM capacity between two strategy groups, we compared the WM span values between the two strategy groups. Since a pair-wise t-test shows no difference in span values across two days [$t(37) = -.197$, $p = .845$], we collapsed the data across days for each strategy group. Next, we used an independent sample t-test to compare the mean visuospatial WM span between the two strategy groups. We found no significant difference in visuospatial WM span between the remember-all (mean span = 5.500, SD = .577) and the remember-subset groups (mean span = 5.526, SD = .716) [$t(36) = -.125$, $p = .901$]. This suggests that the baseline VSTM capacity between these two groups were comparable and that any effect of strategy or tDCS on VSTM performance could not be a result of baseline individual differences in WM capacity between these groups.

3.2. *Encoding strategy impacts VSTM capacity*

To test the influence of the encoding strategy on VSTM performance, we compared the estimates of VSTM capacity and precision between two groups in the sham condition. As shown in Fig. 3.a (red vs blue bars), task strategy affected the capacity of VSTM in the way that is consistent with the findings recently reported by Bengson and Luck (2016). Remember-all strategy significantly increased participants' VSTM capacity in the supra-capacity condition (set size 8) without changing VSTM capacity at lower set sizes (set sizes 4 and 6) or changing VSTM precision in any set size. These results were verified by the following statistical analysis.

The mixed three-way repeated measures ANOVA on VSTM capacity in the sham condition revealed that there was a significant main effect of task strategy, specifically a significant increase in VSTM capacity in the remember-all group compared to the remember-subset group [$F(1, 36) = 6.042$, $p = .019$, $\eta_p^2 = .144$]. Moreover, we also observed a significant main effect of set size [$F(2, 72) = 22.062$, $p < .001$, $\eta_p^2 = .380$], and a significant interaction between set size and task strategy [$F(2, 72) = 4.802$, $p = .011$, $\eta_p^2 = .118$]. Pre-planned pair-wise

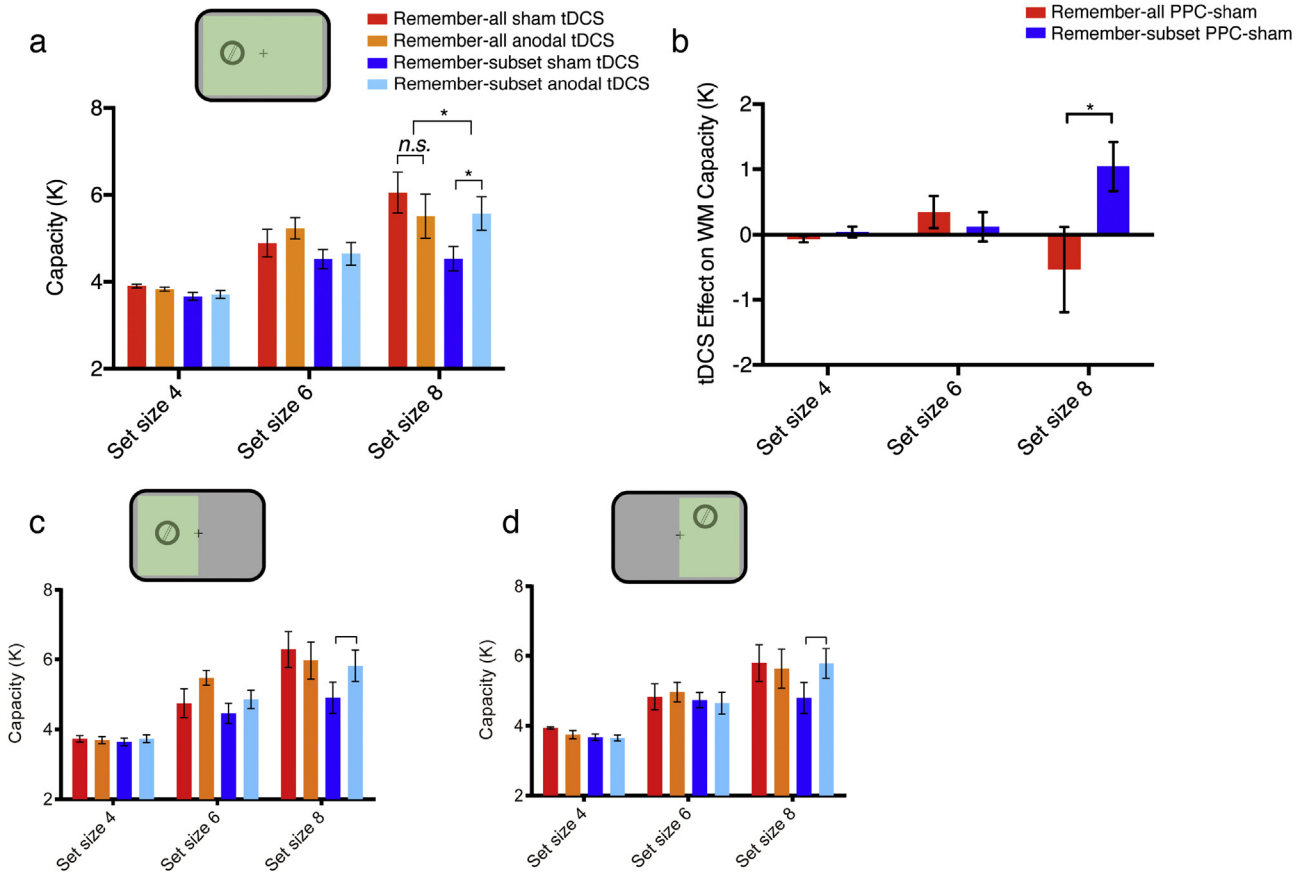

Fig. 3 – The effect of PPC stimulation on VSTM capacity depends on subjects' encoding strategy. (a) The mean values of the VSTM capacity in different set size and stimulation conditions across the remember-all and the remember-subset groups. There was a stimulation effect only for set size 8 in the remember-subset group (compare blue and cyan bars) but not in the remember-all group (compare red and orange bars). (b) The differential capacity values between the actual PPC stimulation and sham shown in (a). Red and blue bars represent remember-all group and remember-subset group, respectively. (c) The mean values of the VSTM capacity in different set size and stimulation conditions across the remember-all and the remember-subset groups when the probed item was presented in the left hemifield of the screen. (d) The mean values of the VSTM capacity in different set size and stimulation conditions across the remember-all and the remember-subset groups when the probed item was presented in the right hemifield of the screen. All error bars indicate the standard error of the mean (SEM). * signs represent *p*'s < .05 (Holm-Bonferroni-corrected). *n.s.* stands for not significant. Red/orange and blue/cyan bars represent the remember-all group and the remember-subset group, respectively.

comparisons across each neighboring set size revealed that VSTM capacity in sham stimulation condition significantly increased from set size 4 to set size 6 [$F(1, 36) = 27.027$, $p < .001$, $\eta_p^2 = .429$], and from set size 6 to set size 8 [$F(1, 36) = 5.635$, $p = .023$, $\eta_p^2 = .135$]. Post-hoc independent-samples *t*-tests revealed that the interaction was driven by a significant increase in VSTM capacity in the remember-all compared to the remember-subset groups for set size 8 [$t(36) = 2.798$, $p = .008$, Cohen's $d = .907$, passing the Holm-Bonferroni threshold of .017], without any significant difference for lower set sizes [$t(36)$'s = 2.321 and .937, p's = .026 and .355, Cohen's d's = .752 and .304 for set sizes 4 and 6, respectively, not passing the Holm-Bonferroni threshold of .025]. Moreover, post-hoc paired-samples *t*-tests revealed that the capacity (K) for remember-all group increased significantly from set size 4 to set size 6 [$t(18) = 3.134$, $p = .006$, Cohen's $d = .991$], and from set size 6 to set size 8 [$t(18) = 2.709$, $p = .014$, Cohen's $d = .665$]. However, the capacity (K) for remember-subset group increased significantly from set size 4 to set size 6 [$t(18) = 5.255$, $p < .001$, Cohen's $d = 1.167$], but stopped increasing from set size 6 to set size 8 [$t(18) = .025$, $p = .980$, Cohen's $d = .006$].

In contrast to the capacity results, the mixed three-way repeated measures ANOVA on VSTM precision (Fig. 4.a; red *vs* blue bars) revealed no main effect of task strategy [$F(1, 36) = .291$, $p = .593$, $\eta_p^2 = .008$], no main effect of set size [$F(2, 72) = .350$, $p = .706$, $\eta_p^2 = .010$], and no interaction between these two independent factors [$F(2, 72) = .468$, $p = .628$, $\eta_p^2 = .013$].

Together, the increase in VSTM capacity at the highest set size in the remember-all compared to the remember-subset group suggests that our manipulation of encoding strategies successfully influenced VSTM performance in the way that closely replicated the earlier findings reported by Bengson and Luck (2016), indicating that subjects in each group followed our task instructions. Moreover, no difference in VSTM capacity and precision at lower set sizes as well as no changes in the WM span reported in the previous section suggests that

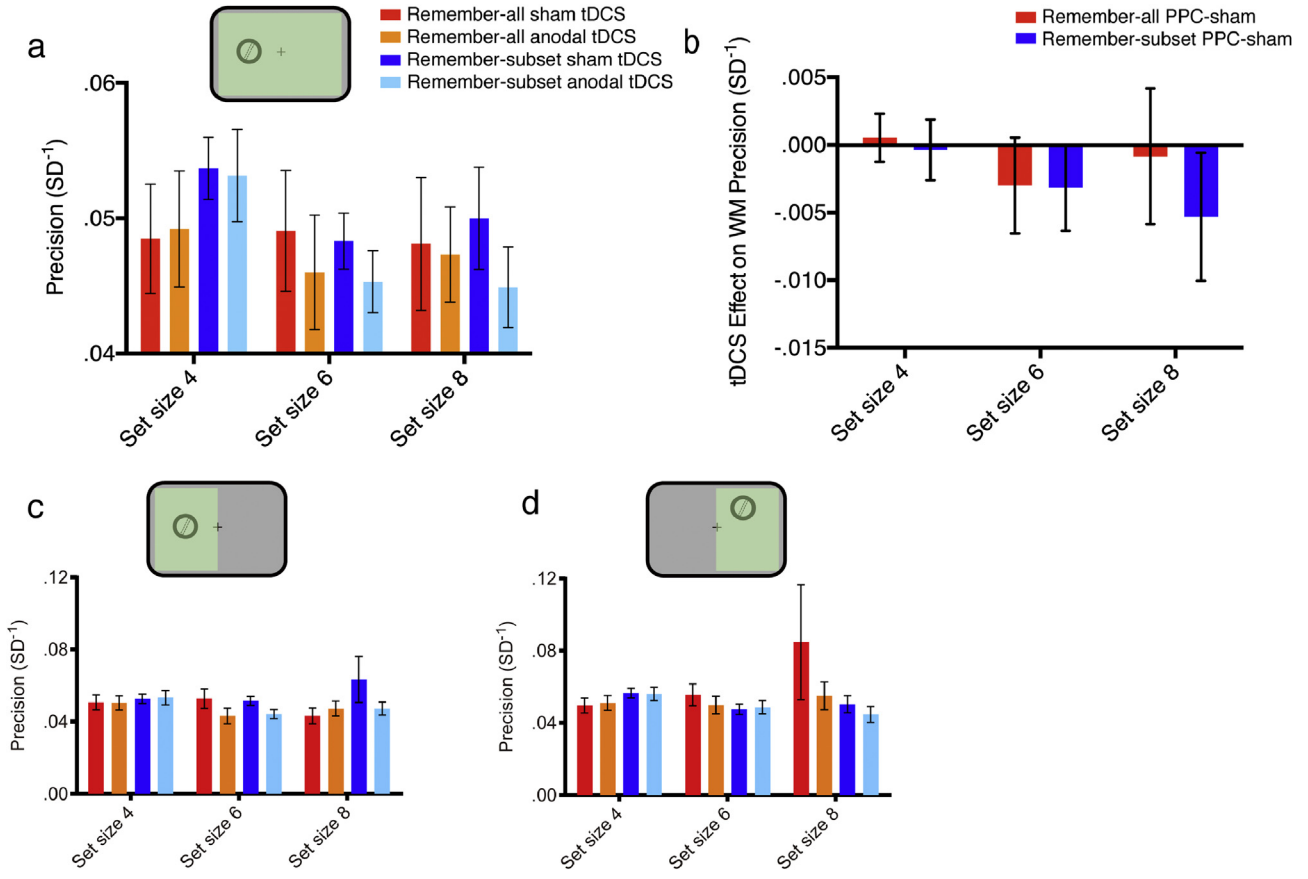

Fig. 4 – The effect of PPC stimulation on VSTM precision. (a) The mean values of the VSTM precision in different set size and stimulation conditions across the remember-all and the remember-subset groups. (b) The differential precision values between the actual PPC stimulation and sham shown in (a). (c) The mean values of the VSTM precision in different set size and stimulation conditions across the remember-all and the remember-subset groups when the probed item was presented in the left hemifield of the screen. (d) The mean values of the VSTM precision in different set size and stimulation conditions across the remember-all and the remember-subset groups when the probed item was presented in the right hemifield of the screen. All error bars indicate the standard error of the mean (SEM). Red/orange and blue/cyan bars represent the remember-all group and the remember-subset group, respectively.

any stimulation effect and its interaction with encoding strategies should not be confounded by individual differences in baseline VSTM functions across the two subject groups.

3.3. PPC stimulation effect on VSTM capacity depends on task strategy

As shown in Fig. 3.a&b, the anodal tDCS over the PPC selectively increased VSTM capacity in the remember-subset group up to the capacity level in the remember-all group in set size 8. Fig. 5 illustrates individual subject's PPC stimulation effects on VSTM capacity in set size 8 for both groups.

The mixed repeated measures ANOVA with a between-subjects factor of task strategy and two within-subjects factors of stimulation type and set size on VSTM capacity showed that there was a marginal significant effect of task strategy [$F(1, 36) = 3.150$, $p = .084$, $\eta_p^2 = .080$], and a significant effect of set size [$F(2, 72) = 44.722$, $p < .001$, $\eta_p^2 = .554$]. However, there was no significant effect of stimulation type [$F(1, 36) = 1.535$, $p = .223$, $\eta_p^2 = .041$]. Pre-planned pair-wise comparisons across each neighboring set size revealed that VSTM capacity significantly increased from set size 4 to set size 6 [$F(1, 36) = 55.903$, $p < .001$, $\eta_p^2 = .608$], and from set size 6 to set size 8 [$F(1, 36) = 12.184$, $p = .001$, $\eta_p^2 = .253$]. The analysis also yielded a significant interaction between stimulation type, task strategy, and set size [$F(2, 72) = 3.721$, $p = .029$, $\eta_p^2 = .94$] as well as a marginal interaction between stimulation type and task strategy [$F(1, 36) = 3.654$, $p = .064$, $\eta_p^2 = .092$]. These interactions were driven by a significant main effect of stimulation type [$F(1, 18) = 8.373$, $p = .010$, $\eta_p^2 = .317$] and a significant interaction between stimulation type and set size on VSTM capacity in the remember-subset group [$F(2, 36) = 4.397$, $p = .020$, $\eta_p^2 = .196$] without a significant stimulation effect [$F(1, 18) = .161$, $p = .693$, $\eta_p^2 = .009$] or its interaction with set size [$F(2, 36) = .216$, $p = .807$, $\eta_p^2 = .012$] in the remember-all group. The follow-up post-hoc paired-sample *t*-tests showed that the interaction between stimulation type and set size in the remember-subset group was driven by the fact that PPC stimulation (relative to sham) significantly enhanced VSTM capacity at set size 8 [$t(18) = 2.767$, $p = .013$, Cohen's $d = .718$, passing the Holm-Bonferroni-corrected threshold of .017], without any stimulation effect at any of the lower set sizes [$t(18)$'s = .520 and .550, $p = .609$ and .589, Cohen's $d = .109$ and .116 for set sizes 4 and 6, respectively, not passing the Holm-Bonferroni-corrected threshold of .025]. Importantly, we found no stimulation effect on VSTM capacity at set size 8 in the remember-all group [$t(18) = .818$, $p = .424$, Cohen's $d = .253$], and the magnitude of the stimulation effect in the remember-subset group was significantly higher than the remember-all group at this set size [$t(36) = 2.090$, $p = .044$, Cohen's $d = .678$] (see Fig. 3b).

Although we observed no significant PPC stimulation effect in the remember-all group, in average there was a reduction in VSTM capacity with PPC stimulation for set size 8 in this group (Fig. 3b). To further test whether the null effect of PPC tDCS on VSTM capacity for the remember-all group was robust or not, we ran an additional Bayes factor analysis. We converted the t value between VSTM capacity of remember-all group in sham and active tDCS condition for set size 8 into a Bayes factor, which indicates the relative likelihood of the null hypothesis versus the alternative hypothesis (Rouder, Speckman, Sun, Morey, & Iverson, 2009). We found that the data are 3.13 times more likely to be observed under the null hypothesis, suggesting that the null effect of PPC tDCS on the capacity parameter of the remember-all group was robust.

Unlike the VSTM capacity results, we found no changes in VSTM precision with stimulation (see Fig. 4.a, b). The mixed repeated measures ANOVA with a between-subjects factor of task strategy and two within-subjects factors of stimulation type and set size on VSTM precision showed that there were no main effect of stimulation type [$F(1, 36) = 2.123$, $p = .154$, $\eta_p^2 = .056$], no main effect of task strategy [$F(1, 36) = .099$, $p = .755$, $\eta_p^2 = .003$], no main effect of set size [$F(2, 72) = 2.349$, $p = .103$, $\eta_p^2 = .061$], no interaction between stimulus type and task strategy [$F(1, 36) = .471$, $p = .497$, $\eta_p^2 = .013$] and no

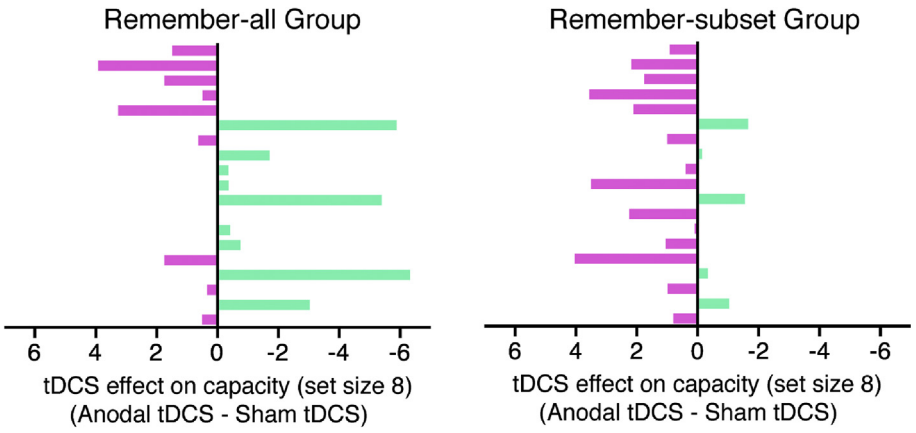

Fig. 5 – Individual subject's PPC stimulation effects on VSTM capacity for set size 8 in the remember-all group (left panels) and the remember-subset group (right panels). Pink and green bars represent better and worse performance induced by PPC stimulation compared to sham.

interaction among stimulus type, task strategy, and set size [$F(2, 72) = .176$, $p = .839$, $\eta_p^2 = .005$].

It is true that in average there was a noticeable reduction in VSTM precision for set size 8 in the remember-subset group though ANOVAs suggest no significant main effect or interaction caused by PPC stimulation on the precision parameter (Fig. 4b). To further test whether the null effect of PPC tDCS on VSTM precision for set size 8 in the remember-subset group was robust, we converted the t value obtained from the pairwise comparison of the VSTM precision values between sham and active stimulation in set size 8 of the remember-subset group into a Bayes factor. We found that the null hypothesis, which predicted no effect of PPC stimulation on the VSTM precision for set size 8 in the remember-subset group, was 4.05 times more likely than the alternative hypothesis, suggesting that the null effect of PPC tDCS on VSTM precision for set size 8 in the remember-subset group was robust.

3.4. *No lateralization of the PPC stimulation effect*

Recent studies exploring the function of the PPC in attentional processes via tDCS have shown that anodal tDCS over the right PPC selectively improved attentional modulations of visual stimuli presented in the contralateral but not in the ipsilateral hemifields. These include inducing spatial bias towards the contralateral hemispace (Sparing et al., 2009), speeding up responses to contralateral targets in the attentional tasks (Bolognini, Olgiati, Rossetti, & Maravita, 2010), and improving spatial reorientation to the targets presented in the contralateral visual field (Roy, Sparing, Fink, & Hesse, 2015). Since attention may facilitate VSTM, the right PPC stimulation may also differentially modulate the memory performance of the targets presented in the contralateral and the ipsilateral visual fields. To examine this possibility, we analyzed VSTM performance for trials with the targets presented in the left and the right visual fields separately. To do so, we computed the recall error separately for trials where the probe was presented in the left and the right hemifields and fit the data with the Standard Mixture model to obtain the capacity and precision values in the left and the right hemifields, respectively. Then, we performed mixed repeated-measures ANOVAs with a between-subjects factor of encoding strategy (remember-all and remember-subset) and other three within-subjects factors of hemifield (left and right), stimulation type (active and sham) and set size (4, 6, and 8) on these capacity and precision values. For the capacity parameter (Fig. 3.c, d), we found no main effect of hemifield [$F(1, 36) = .557$, $p = .460$, $\eta_p^2 = .015$], no interaction between hemifield and stimulation type [$F(1, 36) = .806$, $p = .375$, $\eta_p^2 = .022$] no interaction between hemifield, stimulation type and encoding strategy [$F(1, 36) = .003$, $p = .956$, $\eta_p^2 = .000$], no interaction between hemifield, stimulation type and set size [$F(2, 72) = 1.351$, $p = .265$, $\eta_p^2 = .036$] and no interaction between hemifield, stimulation type, set size and group [$F(2, 72) = .011$, $p = .989$, $\eta_p^2 = .000$]. Similarly, for the precision parameter (Fig. 4.c, d), we found no main effect of hemifield [$F(1, 36) = 1.195$, $p = .282$, $\eta_p^2 = .032$], no interaction between hemifield and stimulation type [$F(1, 36) = .092$, $p = .763$, $\eta_p^2 = .003$] no interaction between hemifield, stimulation type and encoding strategy [$F(1, 36) = 2.163$, $p = .150$, $\eta_p^2 = .057$], no interaction between hemifield, stimulation type and set size [$F(2, 72) = 1.135$, $p = .327$, $\eta_p^2 = .031$] and no interaction between hemifield, stimulation type, set size and group [$F(2, 72) = 2.230$, $p = .115$, $\eta_p^2 = .058$]. Over all, these null lateralization results suggest that it is unlikely that changes in VSTM capacity observed in our study was driven from changes in attentional processes, especially those inducing more spatial bias to the contralateral hemifield.

4. Discussion

In the present study, we applied anodal tDCS over the PPC in human subjects who were instructed to adopt different encoding strategies during a variant of VSTM recall task. We first replicated the recent finding where subjects in the remember-all group outperformed those in the remember-subset group in the most difficult supra-capacity condition (Bengson & Luck, 2016). Moreover, PPC stimulation selectively enhanced VSTM capacity only in the remember-subset group but not in the remember-all group, with the stimulation-induced enhancement in VSTM capacity in the former group reaching the capacity level observed in the latter group. These results suggest that encoding strategy directly impacts VSTM capacity and this influence of encoding strategy mediates the effects of PPC stimulation on VSTM capacity. Therefore, individual difference in encoding strategy may be one of the key factors that contribute to the variability in PPC-stimulation effects reported by past studies (c.f., Horvath et al., 2015; Mancuso et al., 2016).

The present study suggests that the difference in encoding strategy is one of the key factors that may contribute to the influences of individual differences on tDCS effect (i.e., baseline performance, education, age, anatomy, genotype, physiological factors) (see reviews, Ridding & Ziemann, 2010; Li, Uehara, & Hanakawa, 2015; Greenwood, Blumberg, & Scheldrup, 2018). The application of tDCS allows small amounts of direct current (usually .5–2 mA) pass through the skull and scalp into cortical tissue under the stimulated regions. It has been shown in animal models that this type of current causes the depolarization of the membrane potential of pyramidal neurons (Radman, Ramos, Brumberg, & Bikson, 2009). Given this finding, we predicted that the anodal tDCS would depolarize the membrane potentials of pyramidal neurons that lay perpendicularly with the cortical surface within the region of the PPC, as shown in the current density distribution model in Fig. 2.d. Accordingly, this depolarization should increase the neuronal excitability within this cortical region via voltage-dependent ion channels (Nitsche & Paulus, 2000, 2001; Nitsche et al., 2003; for review, see; Polanía, Nitsche, & Ruff, 2018). The stimulation that lasted over 15 min should also lead to changes in synaptic plasticity regulated by NMDA glutamatergic and GABAergic receptors, producing a neuronal excitability effect over hours (Nitsche et al., 2003; Stagg et al., 2009; for review, see; Polanía et al., 2018). Accordingly, this neuronal plasticity should enhance the excitability of the PPC during the VSTM task.

Several past neuropsychological studies in patients have found that right parietal damage causes VSTM deficits (Berryhill & Olson, 2008; De Renzi, Faglioni, & Previdi, 1977; Husain et al., 2001; Mackey, Devinsky, Doyle, Golfinos, & Curtis, 2016; Malhotra et al., 2005; Pisella, Berberovic, & Mattingley, 2004; Ravizza, Behrmann, & Fiez, 2005; Vallar & Coslett, 2018). Moreover, many fMRI and EEG studies have shown that the magnitude of PPC activity tracks the number of items held in VSTM up to its capacity limit (Ikkai et al., 2010; Luria et al., 2016; Todd & Marois, 2004, 2005; Vogel & Machizawa, 2004; Xu & Chun, 2006). According to these findings, we predict that the anodal tDCS increases the excitability of the PPC allowing more objects to be maintained in VSTM for remember-subset group. In contrast to the remember-subset group, subjects who adopted the remember-all strategy might have already exhausted all neuronal resources in the PPC. Therefore, PPC stimulation in this group might lead to no or minimal change in overall excitability of the PPC, compared to sham stimulation. Future studies could use similar behavioral and modeling approaches in combination with neurobiological assessments (e.g., fMRI and EEG) to test the possible neural mechanisms we raised here.

In addition, the selective tDCS effect at set size 8 is consistent with many past studies that observed greater stimulation-induced benefits in VSTM functions at higher difficulty levels (Berryhill, Peterson, Jones, & Stephens, 2014; Jones & Berryhill, 2012; Li et al., 2017; Wang, Itthipuripat, & Ku, 2019). This might be because individuals only need to increase their VSTM capacity to perform better in the higher difficulty levels. Even though both set sizes are over capacity limit, set size 8 is relatively more difficult than set size 6. For the remember-subset group in sham condition, their VSTM capacity increased from set size 4 to set size 6, but stopped increasing from set size 6 to set size 8. This behavior suggests that they could not perform any better than set size 6, therefore PPC-stimulation would be the most effective for set size 8. On the other hand, the visual WM capacity value of the remember-all group significantly increased from set size 4 to set size 6, and from set size 6 to set size 8 ($ps < .014$). This pattern suggests that they could handle all three set sizes, hence no PPC-stimulation-induced effect was observed in this group.

It is worth noting that our tDCS results showed only a selective stimulation effect on VSTM capacity, but not on VSTM precision, consistent with the findings of our recent study (Wang et al., 2019). The null effects of the PPC stimulation on the precision parameter are also consistent with the recent idea that VSTM capacity and precision might depend on distinct neural substrates. Specifically, the PPC may have a more dominant role in supporting the capacity storage for discrete items in VSTM but visual areas in the occipital cortex might relatively perform better at encoding precise representations of sensory information (Bettencourt and Xu, 2015; Xu, 2017, 2018; Ester, Sprague, & Serences, 2015, 2016; Leavitt, Mendoza-Halliday, & Martinez-Trujillo, 2017; Gayet, Paffen, & Van der Stigchel, 2018; Scimeca, Kiyonaga, & D'Esposito, 2018; Zhao, Kuai, Zanto, & Ku, 2020; but see Rademaker, Chunharas, & Serences, 2019).

One may have concerns regarding the robustness of the current results by pointing out that our interpretation relies on one single finding that the stimulation effect only appears in the remember-subset group, only at set size 8, and in the capacity parameter. However, we are convinced that this particular finding is very robust by the following reasons. First, the finding that the stimulation effect only appeared in high set sizes and was only reflected in capacity measurement has already been established by previous studies (Berryhill et al., 2014; Jones & Berryhill, 2012; Li et al., 2017; Wang et al., 2019). Moreover, statistically, this particular result is very robust as we observed a significant interaction between stimulation type and set size with a large effect size ($\eta_p^2 = .317$) in the remember-subset group. It is true that we only found such significant interaction in the remember-subset group but not in the remember-all group; however, that was the main hypothesis that we were testing. Note that we didn't only observe the significant and the null interaction results in the remember-subset and the remember-all groups, respectively, but this group difference was also statistically confirmed by a significant three-way interaction between group, stimulation type, and set size with an extremely large effect size ($\eta_p^2 = .94$). Therefore, we are convinced that this novel finding is very robust. That said, given the novelty of this result, it's worth following up on by future studies.

Interestingly, our results showed no lateralization effect of the right PPC stimulation. This is consistent with results from many past neuroimaging studies demonstrating the functional role of the PPC in VSTM (e.g., Todd & Marois, 2004, 2005; Xu & Chun, 2006). In contrast to these results, recent studies exploring the function of the PPC in attentional processes via tDCS have shown that anodal tDCS over the right PPC selectively improved attentional modulations of visual stimuli presented in the contralateral but not in the ipsilateral hemifields. These include inducing spatial bias towards the contralateral hemispace (Sparing et al., 2009), speeding up responses to contralateral targets in the attentional tasks (Bolognini et al., 2010), and improving spatial reorientation to the targets presented in the contralateral visual field (Roy et al., 2015). Taken together, our null lateralization results suggest that it is unlikely that changes in VSTM capacity observed in our study was driven from changes in attentional processes, especially those inducing more spatial bias to the contralateral hemifield.

The selective tDCS effect on VSTM capacity in the remember-subset group could be due to the PPC-stimulation effect on VSTM capacity which was modulated by the encoding strategy or the PPC-stimulation effect on encoding strategy which in turn influenced VSTM capacity. We think the former one could better explain the current results because the association between the magnitude of the PPC activity and the number of items held in VSTM has been well established by substantial fMRI and EEG studies (Ikkai et al., 2010; Luria et al., 2016; Todd & Marois, 2004, 2005; Vogel & Machizawa, 2004; Xu & Chun, 2006). Therefore, increasing the excitability of the PPC by anodal tDCS might allow more objects to be maintained in VSTM for the remember-subset group, not for subjects in the remember-all group who might have already exhausted all neuronal resources in the PPC. It is possible that the current results may be due to the effect of the PPC-stimulation on encoding strategy itself which in turn

influenced participants' VSTM capacity. However, we have no clear way to tell whether stimulating the PPC would modulate an individual's encoding strategy or not. Moreover, the brain area involved in modulating the encoding strategy during VSTM task is still unknown. Thus, future experiments are needed to address this issue.

While individual differences in VSTM capacity/precision could be a potential confound with the between-groups design, we do not think it likely contributed to the findings we observed here for many reasons. First, all the participants were randomly assigned to the remember-all and the remember-subset groups without any bias. As a result, there were no significant differences in VSTM capacity/precision at lower set sizes. Moreover, prior to stimulation on both days, we had subjects undergo the visuospatial WM span task to obtain an independent measure of their VSTM functions. We found no difference in the visuospatial WM span between the remember-all and the remember-subset groups. Together, these null results suggest that individual differences in baseline VSTM functions should not be a key driver of the stimulation effects we found in the current study.

It is possible that our results could be influenced by subjects knowing which stimulation session they are in by guessing from the different sensation from active and sham stimulation, which were hard to address due to a lack of after-experiment questionnaires. However, we think this is unlikely for many reasons. First, we told subjects prior to the experiment that the stimulation might lead to better or worse performance so that they could not adjust their behavior in a specific direction based on guessing by the sensation from the stimulation. Moreover, even if subjects could guess what session they were in, changes in performance should have generally impact both capacity and precision measures and across all set sizes; there should not be a selective enhancement effect on the capacity value only at set size 8. Thus, we believe that the selective PPC-stimulation effect on the capacity measurement at set size 8 could not be due to subjects knowing which stimulation conditions they were in.

One limitation of the current experiment is not having a control region of the active PPC-stimulation. That is to say, in the present study the interpretation regarding the PPC stimulation effects were based on the difference between sham and active stimulation over the PPC, but not the difference between the PPC and other stimulation sites. That said, according to our recent study using a similar behavioral paradigm and tDCS set-up except that we also used the prefrontal cortex (PFC) as a control site (Wang et al., 2019). We found that stimulating PPC selectively increased the VSTM capacity compared to sham and PFC stimulation. Taken together our past and current findings, we think that the stimulation-induced enhancement in VSTM capacity is specific to the PPC and it is not due to global excitability induced by our tDCS protocols.

Another limitation of our study is that there was no subjective measurement showing how well subjects followed our task strategy instructions. How could we know that they actually followed our task instructions? To ensure that they did so, we gave them the exact verbatim instructions used by a previous study that successfully manipulated VSTM encoding strategy (Bengson & Luck, 2016). As described earlier, this study has recently shown that telling subjects to remember all items in the supra-capacity array helped increase their VSTM capacity as it surpassed the commonly observed capacity limit (~3–4 items) compared to when subjects were not given a specific instruction or when they were told to remember only a subset of those memory items. Moreover, prior to brain stimulation, we trained subjects to employ a given strategy over 100 trials to ensure that they understood the instruction and had substantial amounts of practice in implementing such strategy. Critically, we adopted the similar between-subjects design for strategy manipulation used by Bengson and Luck (2016) to make sure that subjects were not aware of the other strategy so that they would not switch between strategies randomly across trials or choose one that they preferred. It is clear from our results that the participants followed the encoding-strategy instruction because the remember-all group had much higher capacity, indicating that they could hold more items in their VSTM compared to the remember-subset group. If the remember-all group did not follow the instruction, we would have seen similar VSTM performance across the two groups in the sham condition.

Note that Bengson and Luck (2016) found remember-all groups outperformed remember-subset groups at all set sizes, with numerically much larger differences at set sizes 6 and 8 than at set size 4. However, the current experiment only revealed a significant higher capacity in remember-all groups than remember-subset groups at set size 8. Despite a seemingly different pattern, we think that the group effects at higher set sizes are actually qualitatively similar across our study and the recent study reported by Bengson and Luck (2016). That is to say, in average the capacity values in the remember-all group were higher than those in the remember-subset group for all set sizes (a significant main effect of strategy), with a relatively higher magnitude for higher set sizes (set sizes 6 and 8 in Bengson and Luck's experiment, while set size 8 in the current experiment). We think the lack of significant group difference for set size 6 in the current experiment could be due to our data being more variable than those reported by Bengson and Luck (2016). And this could result from the fact that we use the recall task instead of the delay match-to-sample task, which generally needs a lot more trials to obtain stable fit capacity and precision parameters.

5. Conclusion

In conclusion, we showed that the PPC stimulation effects on VSTM capacity varied depending on the encoding strategy subjects employed during the VSTM recall task. In particular, subjects in the remember-subset group who performed generally worse than subjects in the remember-all group gained more benefit from the actual PPC stimulation (compared to sham) than subjects in the latter group. This finding suggests that the PPC stimulation effects on VSTM performance are mediated by individual difference in VSTM capacity caused by difference in encoding strategy. Ultimately, our results help reconcile the discrepancy between inconsistent PPC stimulation effects on VSTM function

reported by past studies and emphasize that difference in task strategy should be carefully taken into account when studying the role of neocortex in supporting VSTM function.

Contributions

S.W. and Y.K. designed research; S.W. performed research; Y.K. supervised research; S.W. and S.I. analyzed data; S.W. and S.I. wrote the first draft of the paper; S.W., S.I., and Y.K. edited the paper.

Open practices

The study in this article earned Open Materials and Open Data badges for transparent practices. Materials and data for the study are available at https://osf.io/ugdh9.

Declaration of Competing Interest

The authors declare no competing financial interests.

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